

Refer to: Gamsu G, Peters DR, Hess D, et al: Isolated right upper lobe pulmonary edema. *West J Med* 135:151-154, Aug 1981

Isolated Right Upper Lobe Pulmonary Edema

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ATYPICAL ACCUMULATION of fluid within the lung may produce unilateral or dominantly lobar pulmonary edema.¹ Unilateral pulmonary edema has been attributed to numerous causes including rapid evacuation of a pneumothorax, systemic-to-pulmonary artery shunt for cyanotic congenital heart disease, rapid thoracentesis and renal transplantation.²⁻⁷ Pulmonary edema localized to one lobe is exceedingly rare and is usually present in the right upper lobe.⁸ We present two cases of lobar edema and possible mechanisms for the localization of the fluid.

Reports of Cases

CASE 1. A 67-year-old woman with adult-onset diabetes who had had one previous episode of myocardial infarction was admitted to a local hospital because of an anterior myocardial infarction. A week after discharge she returned because of increasing shortness of breath and epigastric pain. On readmission, a physical examination of the patient disclosed moderate pedal edema and rales at the lung bases; no heart murmurs were heard. Laboratory values for creatinine phosphokinase (CPK), serum aspartate aminotransferase and lactic dehydrogenase (LDH) were within normal limits. Findings on a chest roentgenogram and a perfusion lung scan showed no abnormalities. Her electrocardiogram was unchanged from previous studies. A week later, however, a repeat x-ray study of the chest showed right upper lobe consolidation, and the patient was transferred to

ABBREVIATIONS USED IN TEXT

BUN = blood urea nitrogen
CPK = creatinine phosphokinase
LDH = lactic dehydrogenase
Paco₂ = partial pressure of carbon dioxide (arterial)
Pao₂ = partial pressure of oxygen (arterial)

our institution for further evaluation. On admission, she appeared acutely ill. Her blood pressure was 170 mm of mercury diastolic, pulse rate was 120 and respiratory rate 36 per minute; temperature was 37.5°C (99.5°F). Jugular vein distension to 16 cm and pronounced pedal edema were present. A soft S₁, a loud P₂, and an S₃ gallop were heard. A grade 3/6 holosystolic murmur was audible at the apex of the heart and radiated to the left axilla. The lung fields demonstrated dullness at both bases and rales up to scapulae. Arterial blood gases exhibited a partial pressure of oxygen (Pao₂) of 95 mm of mercury, a slightly low partial pressure of carbon dioxide (Paco₂) and a serum pH of 7.39.

Laboratory values for serum bicarbonate, serum electrolytes, hemoglobin and hematocrit were all within normal limits. Blood urea nitrogen (BUN) was 56 mg per dl and creatinine 2.3 mg per dl. A leukocyte count was 14,200 per cu mm, with a normal differential count.

A roentgenogram of the chest showed patchy consolidation in the right upper lobe and small bilateral pleural effusions (Figure 1). A tentative diagnosis of pneumonia was made, although pulmonary hemorrhage, infarction and contusion were considered in the radiographic differential diagnosis. Microscopic examination of sputum and transtracheal aspirate specimens detected no polymorphonuclear leukocytes or organisms. Pleural fluid, obtained by thoracentesis, was a transudate with no organisms. Cultures of sputum, transtracheal aspirate and pleural fluid grew no organisms. A Swan-Ganz thermodilution catheter was positioned in the right pulmonary artery and subsequently wedged in the right upper lobe consolidation. The right atrial pressure was 17 mm of mercury and pulmonary artery pressure 78/40 mm of mercury (mean 59 mm of mercury). Pulmonary capillary wedge pressure was 44, with a "V" wave of 65 mm of mercury. Cardiac output was 3.1 liters per minute, with an arteriovenous oxygen difference of 9.9 ml per dl and a systemic vascular resistance of 2,323 dyne•sec•cm⁻⁵. CPK and LDH isoenzyme values suggested acute myocardial in-

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Submitted, revised, September 19, 1980.

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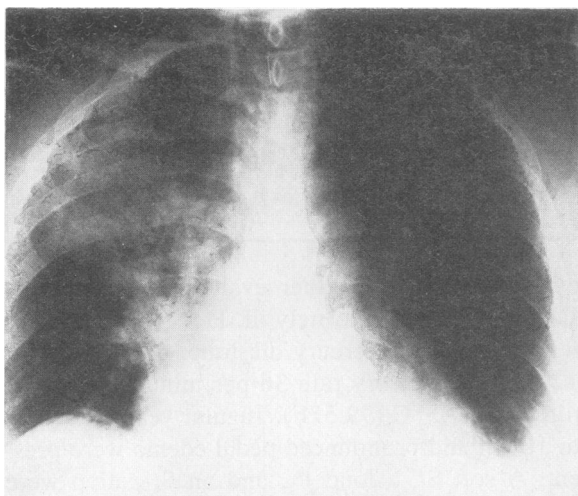


Figure 1.—(Case 1) Frontal portable roentgenogram of the chest shows dense, right upper lobe consolidation. The remainder of the lung fields is essentially clear.

farcion. A clinical diagnosis was made of papillary muscle dysfunction leading to acute mitral regurgitation.

The patient was treated by afterload reduction with nitroprusside and showed improvement in cardiac output and a pronounced decrease in pulmonary capillary wedge pressure. The right upper lobe consolidation cleared in 24 hours, indicating the correct diagnosis of lobar pulmonary edema. Subsequent cardiac catheterization showed moderate to substantial mitral regurgitation, apico-inferior myocardial hypokinesis and diffuse coronary artery disease involving three vessels.

CASE 2. A 26-year-old woman was admitted to a local hospital for hypertension and slow thought processes. Five years before, hemodialysis had been initiated for end-stage renal failure caused by chronic glomerulonephritis and hypertension. She had had a successful cadaver renal transplantation two years later. Her maintenance medications included 75 mg per day of azathioprine and 30 mg per day of prednisone. Two weeks before admission she began to gain weight and peripheral edema developed. When admitted to the hospital, she had tenderness over the site of her renal graft and elevated BUN and serum creatinine levels. A diagnosis of allograft rejection was made and the patient was treated with 60 mg per day of prednisone and a continuance of 75 mg of azathioprine per day. She did not improve and after a week was transferred to our institution for placement of a vascular shunt for maintenance hemodialysis.

At the time of transfer, her mentation was still sluggish, but she had no chest pain, cough or

dyspnea. She was 15 lb (6.8 kg) over her usual weight, peripheral edema was pronounced, and blood pressure was 190/120 mm of mercury. The

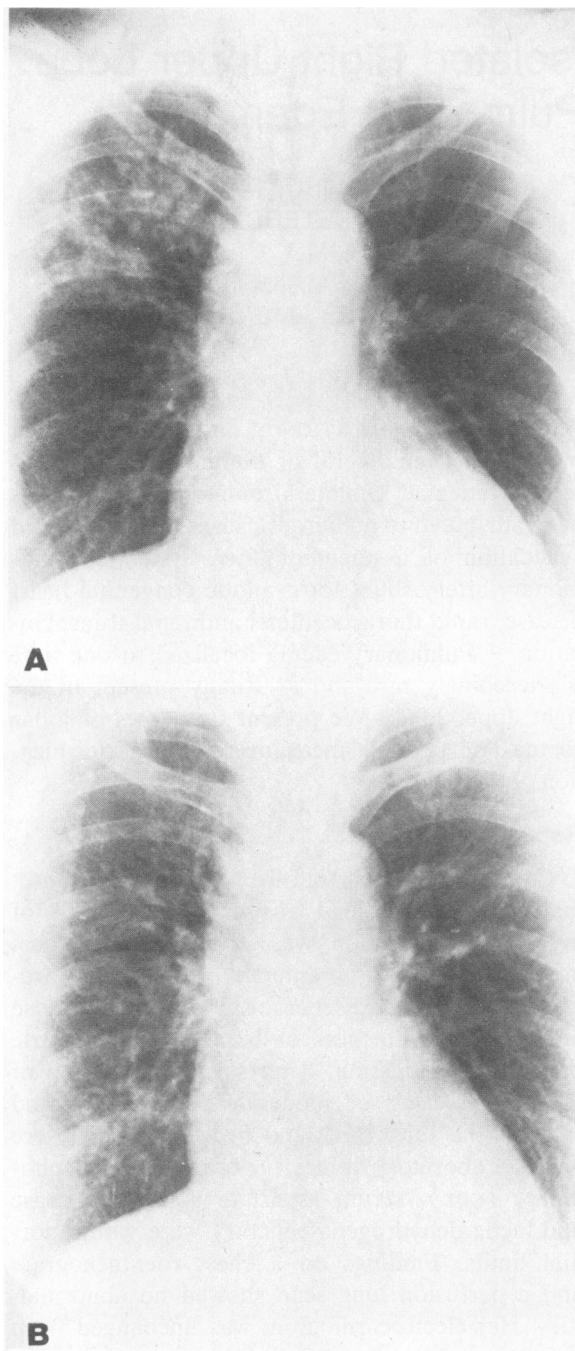


Figure 2.—(Case 2) **A**, Frontal roentgenogram of the chest at the time the patient was transferred shows patchy consolidation in most of the right upper lobe. The remainder of the lung fields is clear and the pulmonary vessels are of normal caliber. Cardiomegaly is visible. **B**, A roentgenogram done three days later shows the right upper lobe consolidation has completely resolved. The pulmonary vessels and cardiac size are unchanged.

lung fields were clear to auscultation. Cardiac auscultation showed that S_1 and S_2 were normal; a soft systolic murmur and an S_4 but no S_3 gallop were audible. BUN was 204, creatinine 12.7 and serum albumin 3.0 mg per dl. A leukocyte count was 6,400 per cu mm; Pao_2 was 54 and $Paco_2$ was 38 mm of mercury; pH was 7.37.

An earlier roentgenogram of the chest had shown moderate cardiomegaly with clear lung fields; however, another roentgenogram, made when the patient arrived at our institution, showed fluffy air space consolidation involving only the right upper lobe (Figure 2A). Cardiomegaly and minimally blunted costophrenic angles were also visible. The tentative clinical and radiographic diagnosis was pneumonia or pulmonary hemorrhage.

Because sputum cultures grew no pathogenic organism and tuberculin and fungal skin tests had been negative, antibiotic drugs were not given. Fiberoptic bronchoscopy was planned for the day following the x-ray study of the chest. In the interim, the patient underwent hemodialysis and lost 2.2 kg. The Pao_2 rose to 74 mm of mercury and pH increased to 7.43. Another roentgenogram showed partial clearing of the right upper lobe consolidation, and bronchoscopy was deferred. On the third day after admission, the patient again underwent hemodialysis and lost an additional 2.2 kg. A chest roentgenogram showed complete clearing of the right upper lobe consolidation (Figure 2B), indicating the correct diagnosis of lobar pulmonary edema.

Discussion

The distribution of pulmonary edema is determined by the hydrostatic forces across the pulmonary microcirculation and by the permeability of the pulmonary capillary bed. Edema fluid usually accumulates at the lung bases. Unilateral pulmonary edema can occur when local forces predominate or when the pulmonary vasculature or pulmonary parenchyma is abnormal (Table 1).⁹

Acute consolidation of the right upper lobe is usually due to pneumonia or obstruction of the lobar bronchus. Occasionally, bleeding in the lung can produce acute lobar consolidation. Pulmonary edema limited to the right upper lobe is rare.⁸

In our first patient, right upper lobe consolidation occurred shortly after an acute myocardial infarction with papillary muscle dysfunction and severe acute mitral regurgitation. A retrograde flow of blood due to the regurgitation can be

TABLE 1.—*Differential Diagnosis of Unilateral Pulmonary Edema*

Gravity
Rapid evacuation of a pneumothorax
Rapid thoracentesis
Aspiration of gastric acid and other materials
Contralateral chronic obstructive pulmonary disease associated with left heart failure
Contralateral obliterative bronchiolitis associated with left heart failure
Acute left heart failure and associated pneumothorax (protective effect)
Systemic-to-pulmonary artery shunt for cyanotic congenital heart disease
Bronchial obstruction (drowned lung)
Renal transplantation

directed across the left atrium toward the orifices of the right upper lobe pulmonary veins.¹⁰ We think that this backflow, or backpressure, into the right upper lobe pulmonary veins could have resulted in a focal increase in pulmonary venous pressure. Pulmonary wedge pressure obtained in the right upper lobe at the time of the pulmonary consolidation was 44 mm of mercury with a "V" wave of 65 mm of mercury, reflecting mitral incompetence. Therapy by afterload reduction resulted in the rapid clearing of the consolidation. In a patient with acute mitral regurgitation, the presence of right upper lobe consolidation may represent focal pulmonary edema resulting from local elevation in pulmonary venous pressure. The radiographic features of left heart failure should be carefully sought; they include septal lines, an increase in the caliber of the upper lobe vessels and peribronchial cuffing.

The second case is less easily explained by focal hemodynamic effects. Although the patient did have a soft systolic murmur, she had no definitive evidence of a myocardial infarction or mitral regurgitation. Pulmonary vascular pressures were not measured. Local abnormal capillary permeability may have been present. The patient was in renal failure and "uremic pneumonitis" may have produced focal consolidation. This poorly understood condition may be a form of focal pulmonary edema.

Pulmonary symptoms, especially in an immunosuppressed patient, are often not helpful in distinguishing pneumonia from edema.¹¹ The possibility of an infectious cause for the lobar consolidation was initially considered in both patients, but rapid clearing occurred without anti-

microbial therapy. Failure to consider edema may lead to unnecessary investigation and important delay in initiating therapy.

Summary

Two cases of right upper lobe pulmonary edema with focal lung consolidation are presented. In both patients, the initial clinical and radiographic differential diagnosis was pneumonia, infarction or pulmonary hemorrhage. In one of the two patients, acute mitral regurgitation from papillary muscle dysfunction was probably responsible for the focal pulmonary edema. In the other patient, "uremic pneumonitis" may have caused the focal consolidation. In both patients the consolidation cleared rapidly without antimicrobial therapy.

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Refer to: Clark OH, Gooding GAW, Ljung BM: Locating a parathyroid adenoma by ultrasonography and aspiration biopsy cytology. *West J Med* 135:154-158, Aug 1981

Locating a Parathyroid Adenoma by Ultrasonography and Aspiration Biopsy Cytology

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ULTRASONOGRAPHY is being used to show the precise location of parathyroid tumors before operation in patients with primary hyperparathyroidism.¹⁻³ One of the problems with this technique is that ultrasonography cannot distinguish between a parathyroid tumor and other lesions, such as thyroid nodules and lymph nodes, situated in the same locations.⁴ False-positive diagnoses are, therefore, common. False-negative diagnoses

may also occur because of the inability to localize some small parathyroid tumors. This report concerns a patient with persistent hypercalcemia. His case presented numerous diagnostic difficulties, and eventually it was documented that the patient had at least six parathyroid glands, two being adenomatous. An elusive second adenoma was eventually localized by ultrasonography and confirmed by aspiration biopsy cytology. To our knowledge this is the first report of locating a nonpalpable parathyroid adenoma by such a combination of procedures. Because hyperparathyroidism is a common disorder, occurring in 1 in 700 persons and usually a result of a solitary parathyroid adenoma, the combined use of ultrasonography and aspiration biopsy cytology may prove to be valuable in selected patients with hyperparathyroidism.⁵⁻⁷

Report of a Case

A 70-year-old man had a radical cystectomy and pelvic node dissection with ileal loop diversion five years previously for a grade 2 to 3 transitional cell carcinoma of the bladder with squamous metaplasia. None of 13 nodes were involved. At that time, his calcium level measured 11.1 mg per dl and phosphorus 2.2 mg per dl. A year later he was without evidence of recurrent tumor. The diagnosis of primary hyperparathyroidism was considered, however, because of persistent hypercalcemia in a hypertensive patient with symptoms of polydipsia and constipation. The

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Supported in part by the Medical Research Service of the Veterans Administration.

Submitted, revised, November 21, 1980.

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